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TECHNICAL MEMORANDUM NO. 5
HUMAN HEALTH RISK ASSESSMENT
TOXICITY ASSESSMENT
OPERABLE UNIT 3

ROCKY FLATS ENVIRONMENTAL TECHNOLOGY SITE

U.S. DEPARTMENT OF ENERGY
ROCKY FLATS ENVIRONMENTAL TECHNOLOGY SITE
GOLDEN, COLORADO

ENVIRONMENTAL RESTORATION PROGRAM

OCTOBER 10, 1994

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ORIA	(EPA) Office of Radiation and Indoor Air
OU 3	Operable Unit 3
pCi/g	picocuries per gram
RAGS	(EPA) Risk Assessment Guidance for Superfund
RCRA	Resource Conservation and Recovery Act
rem	roentgen equivalent man
RfD	reference dose
RFI	RCRA facility investigation
RFETS	Rocky Flats Environmental Technology Site
RI	remedial investigation (CERCLA)
risk/pCi	risk per picocurie
risk/rem	risk per roentgen equivalent man
Sv	sieverts
TM	technical memorandum
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WL	working level
WLM	working-level month

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1.0 INTRODUCTION

The purpose of this Toxicity Assessment Technical Memorandum (TM) No. 5 is to present the toxicity assessment and relevant toxicity constants for the two chemicals of concern (COCs) identified in the Human Health Risk Assessment (HHRA) Chemicals of Concern Identification TM No. 4. This TM supports the HHRA for Operable Unit 3 (OU 3) located adjacent to the Rocky Flats Environmental Technology Site (RFETS). The HHRA is being conducted in accordance with the requirement set forth in the 1991 Federal Facility Agreement and Consent Order (Interagency Agreement [IAG]) signed in 1991 (IAG, 1991). OU 3 consists of the following Individual Hazardous Substances Sites (IHSSs):

- IHSS 199: Contamination of Soils
- IHSS 200: Great Western Reservoir
- IHSS 201: Standley Lake
- IHSS 202: Mower Reservoir

The HHRA will assess potential human health risks for exposure of receptors to the COCs under current land use and likely future land-use conditions, assuming no remedial action occurs at OU 3. The HHRA will be included in the Resource Conservation and Recovery Act (RCRA) Facility Investigation/Remedial Investigation (RFI/RI) report for OU 3. The RFI/RI is being conducted pursuant to the U.S. Department of Energy (DOE), the U.S. Environmental Protection Agency (EPA), the State of Colorado Department of Public Health and the Environment (CDPHE), and the IAG (IAG, 1991).

The toxicity assessment in the OU 3 risk assessment involves assessing the potential for americium-241 (^{241}Am) and plutonium-239/240 (^{239,240}Pu) to cause adverse health effects in exposed individuals. A brief discussion of the potential adverse health effects associated with

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exposure to these radionuclides and information on dose-response relationships are presented in this TM. The toxicity assessment contains two components:

1.   Hazard Identification, which is the process of evaluating the adverse human health effects, if any, that may result from exposure to COCs.

2.   Dose-response evaluation, which quantitatively examines the relationship between the level of exposure and the occurrence of adverse health effects in an exposed population. Dose-response relationships, which are expressed as quantitative toxicity constants, are summarized.

The assessment of risks associated with exposure to ionizing radiation is similar in some ways to the assessment of risks associated with chemical carcinogens. Like carcinogenic chemical risks, radiological risks are usually expressed as an increased probability of cancer. However, radiological risks have historically been expressed as the increased probability of induction of a fatal cancer, while chemical risks are usually expressed as the increased probability of cancer incidence. The risk characterization will express radiological risks as increased risk of total cancer incidence, in accordance with EPA methods outlined in RAGS.

Another difference between chemical and radiological risk assessment methods lies in the use of radiation dose equivalent as the primary expression of harm from exposure to radiation. Radiation risks are often calculated by first determining the dose equivalent received (in rems) and then applying a factor that converts dose equivalent to risk. In chemical risk assessments, intake of chemicals (usually expressed in mg/kg-day) is converted to risk, using an intake to risk conversion factor or slope factor (SF). The intake-to-risk approach will be used to determine radiological risks. However, effective dose equivalent values will also be calculated in the risk characterization for comparison to standards.

Toxicity constants in the form of slope factors and effective dose equivalent factors will be used in the HHRA to evaluate potential adverse effects from exposure to site-related chemicals.

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In this TM, chronic toxicity constants are presented, since a goal of the HHRA is to determine whether long-term exposure to site-related COCs is expected to cause adverse health effects in exposed individuals.

To assist in understanding the following discussions, several common radiological terms need explanation. The degree of damage from radiation in biological systems varies in proportion to how much energy is transferred to the tissue over a linear track length by the radiation. This concept is referred to as linear energy transfer (LET). High-LET radiation causes a high degree of ionization by depositing a large amount of energy over a very short distance, thus, potentially producing significantly greater biological damage than low-LET radiation. Alpha particles are the most common example of high-LET radiation. Both ^{241}Am and ^{239,240}Pu are alpha emitters. Low-LET radiation deposits energy over a much longer range and creates less densely ionized regions. Beta particles, gamma rays, and X-rays are examples of low-LET radiation.

Section 2.0
HAZARD IDENTIFICATION

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2.0 HAZARD IDENTIFICATION

EPA classifies all radionuclides as human carcinogens (Group A), based on their property of emitting ionizing radiation and on the extensive weight of evidence provided by epidemiological studies of radiogenic cancers in humans (EPA, 1993; EPA, 1994). At Superfund radiation sites, EPA generally evaluates potential human health risks based on the radiotoxicity, i.e., adverse health effects caused by ionizing radiation, rather than on the chemical toxicity, of each radionuclide present (EPA, 1993).

The effects of exposure to ionizing radiation fall into three general categories: 1) carcinogenic effects; 2) mutagenic (genetic) effects; and 3) teratogenic effects. For this assessment, only the effects of exposure to low levels of ionizing radiation are evaluated.

In the following subsections, the biological damage mechanisms of ionizing radiation are described as well as the carcinogenic, mutagenic (genetic), and teratogenic effects. Section 2.2 presents the rationale for using total cancer incidence as the basis for assessing radiation risks to receptors.

2.1 BIOLOGICAL DAMAGE MECHANISMS

Radiation produces damage in biological systems through ionization of molecules. Damage may occur directly, as when a chromosome breaks into smaller pieces after absorption of energy from radiation. Damage may also occur indirectly, through ionization of water molecules to produce highly reactive free radicals. The free radicals may react with other cellular compounds and cause damage through oxidation reactions.

The biological effects of radiation are classified as either nonstochastic or stochastic effects. Nonstochastic effects are effects that occur only after a minimum (threshold) dose has been received. Examples of nonstochastic effects include reddening of the skin (erythema) and cataracts. Nonstochastic effects are principally associated with high levels of radiation

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exposure (>10 roentgen equivalent man [rem]). A rem is a unit of "dose equivalent" used in radiation protection to measure the amount of damage to human tissue from a dose of ionizing radiation. It is highly unlikely that receptors at OU 3 could ever receive radiation doses that would cause nonstochastic effects.

Stochastic effects are those for which the probability of occurrence increases with the cumulative dose. For stochastic effects, there is no "threshold" dose below which effects do not occur. The stochastic effects associated with low levels of radiation exposure include cancer, genetic effects, and teratogenic effects.

2.2 CARCINOGENIC EFFECTS

Ionizing radiation has been demonstrated to induce human cancer. A great deal of data exist correlating high exposures of radiation to cancer induction in humans. In general, scientists agree that the probability of cancer increases with dose, but scientists continue to debate which dose-response model most accurately predicts the effects of low-level radiation exposure. Current radiation-protection standards are based on the idea that each increment of radiation exposure causes a linear increase in the risk of cancer (the linear nonthreshold hypothesis).

The Biological Effects of Ionizing Radiation (BEIR) V Committee of the National Academy of Science (NAS) recently completed a study entitled Health Effects of Exposure to Low Levels of Ionizing Radiation (otherwise known as the BEIR V Report) (NAS, 1990). The study included information from the continuing epidemiological studies of the Japanese survivors of the atomic bomb and of radiotherapy patients treated for cancer. The BEIR V Committee concluded that the linear nonthreshold dose-response model most accurately predicts the increased risk of most forms of cancer that develop from exposure to low doses of radiation. The BEIR V Committee also increased the cancer risk estimates for radiation exposure from the 1980 BEIR III Report (NAS, 1980) by a factor of 3 to 4, based primarily on results of studies that

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reevaluated the actual radiation doses received by the Japanese survivors of the atomic bomb. However, EPA does not currently use the BEIR V study in evaluating dose and risk assessment.

EPA recently finished evaluating the cancer risk from radiation exposure as part of the safety analysis for radionuclide standards for atmospheric releases (known as National Emission Standards for Hazardous Air Pollutants [NESHAP] (EPA, 1989b). Table 2-1 includes a summary of the current risk estimates used by EPA for cancer induction and cancer mortality from radiation exposure based on BEIR III. These risk estimates are in terms of the excess cancer induction and excess cancer deaths expected in a population of 1 million people, each person exposed to one rad. A rad is defined by the International Commission on Radiation Units and Measurements (ICRU) as the amount, or dose, of ionizing radiation absorbed by any material, such as human tissue. Radiation absorbed dose is expressed as energy per unit mass. One rad is equivalent to 100 ergs of energy absorbed by one gram of absorbing material. The use of these risk estimates in the risk assessment is explained in Section 3.0 of this TM.

2.3 MUTAGENIC (GENETIC) EFFECTS

Radiation can cause damage to cells by changing the number, structure, or genetic content of the genes and chromosomes in the cell nucleus (NAS, 1972, 1980). These heritable radiation effects are classified as either gene mutations or chromosome aberrations. Gene mutations and chromosome aberrations may occur in either somatic (body) or germ (reproductive) cells. When the mutation or aberration occurs in a somatic cell, the damage is expressed in the exposed individual. For somatic-cell mutations, the worst consequence of the damage is cancer induction. When the mutation or aberration occurs in a germ cell, the resulting damage may be expressed in the descendants of the exposed individual.

Followup epidemiological studies of human populations exposed to low doses of radiation have not shown conclusive evidence of heritable effects that are due to radiation exposure. Most scientists agree, however, that these effects may be occurring in numbers so low that they are

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TABLE 2-1

SUMMARY OF CURRENT EPA-RECOMMENDED RADIATION RISK FACTORS

Risk	Significant Exposure Period	Risk Factor (Effect/10⁶ Rad)	
		Nominal	Range
Low-Linear Energy Transfer			
Carcinogenic effects			
Fatal cancers			
All cancers	Lifetime	390	120-1,200
Genetic effects	Lifetime	620	190-1,900
Severe hereditary defects, all generations	30-year reproductive generation		
Teratogenic effects			
Severe mental retardation		260	60-1,100
Malformation	Weeks 8 to 15 of gestation	4,000	
Preimplantation loss	Weeks 2 to 8 of gestation	5,000	2,500-5,500
	Weeks 0 to 2 of gestation	10,000	--
High-Linear Energy Transfer			--
Carcinogenic effects			
Fatal cancers			
All cancers	Lifetime	3,100	960-9,600
Genetic effects	Lifetime	5,000	1,500-15,000
Severe hereditary defects, all generations	30-year reproductive generation	690	160-2,900

Source: EPA, 1989b.

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not detectable in the study populations. Because of the lack of conclusive human data, animal studies are used to determine risk factors for heritable effects in humans.

The results of extensive animal studies have shown that radiation increases the spontaneous, or natural, mutation rate. No new types of mutations have been attributed to radiation exposure. Estimates based on extrapolation from these animal studies are that at least 100 rem of low-dose-rate, low-LET radiation are required to double the spontaneous mutation rate in humans. Current human dose-response models, however, assume that the probability of genetic damage increases linearly with radiation dose, and that there is no evidence of a "threshold" dose for initiating heritable damage to germ cells (EPA, 1989a).

2.4 TERATOGENIC EFFECTS

Relatively high doses of radiation exposure have been shown to produce abnormalities in animals and humans exposed in utero (Brent, 1980). The effects of radiation exposure to the fetus vary with the stage of gestation. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has developed quantitative risk estimates for effects of prenatal irradiation (primarily mental retardation) over the different stages of pregnancy. Possible risks of fetal radiation exposure include mental retardation, development of fatal cancer after birth, malformation, and preimplantation loss or spontaneous abortion.

2.5 SUMMARY

Cancer induction through exposure to low levels of radiation constitutes the most significant potential consequence of exposure. The risks of heritable effects from radiation exposure are much lower than cancer induction for the first few generations. Carcinogenic effects can be induced at any point during a lifetime. However, exposures must occur during a specific period during gestation for the risks of effects on the developing fetus to be significant. In most cases, the cumulative risk of cancer is much higher than the risk of fetal effects or genetic

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effects. For these reasons, cancer induction is used as the basis for assessing the radiation

risks to receptors.

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3.0 DOSE-RESPONSE EVALUATION

The method used for this radiological risk assessment conforms to the guidelines outlined in Chapter 10 of Risk Assessment Guidance for Superfund, Volume I: Health Evaluation Manual, Part A (EPA, 1989a). In accordance with EPA guidance, the risk associated with radiation exposure is evaluated by using age-averaged slope factors that represent lifetime excess cancer incidence per unit of intake for each radionuclide. These factors are tabulated as part of the Health Effects Assessment Summary Tables (HEAST) documentation (EPA, 1994).

This method differs from the historical method of determining risk from radiation exposure, whereby radiological risks are expressed as the probability of the induction of fatal cancer. The historical method and associated risk factors were developed for regulating occupational exposures and may not be appropriate for determining the risks of radiation exposure for the general public. The primary differences between the HEAST slope factors and the historical risk factors are:

- The ingestion and inhalation slope factors from HEAST are best estimates of total (fatal and nonfatal) incidence of cancer (HEAST, 1994). External slope factors are best estimates of lifetime excess cancer incidence. Most historical radiation slope factors are based on fatal cancer incidence.

- The ingestion and inhalation slope factors from HEAST are used to evaluate risk based on the intake of radioactive materials (risk/pCi). External slope factors are expressed as risk/year per pCi/gram of soil or risk/year per Bq/gram soil. The historical slope factors determine risk based on radiation dose (risk/rem).

The results of the HEAST (intake-based) methodology are used to develop estimates of the risk of total cancer incidence. Depending upon the type of cancer, the cancer-incidence risk may range from approximately one to three times the corresponding fatal-cancer risk for a given

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pathway. Table 3-1 lists the internal (ingestion and inhalation) and external slope factors for
COCs.

EPA's Office of Radiation and Indoor Air (ORIA) calculates radionuclide slope factors using
health-effects data, and dose and risk models from a number of national and international
scientific advisory commissions and organizations, including the NAS, the National Council on
Radiation Protection and Measurement (NCRP), UNSCEAR, and the International Commission on
Radiological Protection (ICRP). Radionuclide slope factors are calculated for each radionuclide
individually, based on its unique chemical, metabolic, and radioactive properties.

The internal, ingestion and inhalation, slope factors account for:

- The amount of radionuclide transported into the bloodstream from either the
  gastrointestinal (GI) tract following ingestion, or from the lungs following
  inhalation

- The ingrowth and decay of radioactive progeny produced within the body
  subsequent to intake

- The distribution and retention of each radionuclide (and its associated progeny,
  if appropriate) in body tissues and organs

- The radiation dose delivered to body tissues and organs from the radionuclide
  (and its associated progeny, if appropriate)

- The sex, age, and organ-specific risk factors over the lifetime of exposure

The slope factors are the average risk per unit intake or exposure for an individual in a
stationary population with vital statistics (mortality rates) typical of the United States in 1970.
Radionuclide ingestion and inhalation slope factors are not expressed as a function of body

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TABLE 3-1

TOXICITY CONSTANTS
CARCINOGENIC SLOPE FACTORS[a] FOR ^{241}Am AND ^{239,240}Pu

		Slope Factors		
Radionuclide	Radioactive Half-Life[b]	Ingestion (risk/pCi)[b]	Inhalation (risk/pCi)[b]	External (Risk/year per pCi/g soil)[b]
^{241}Am	432 years	2.4×10^{-10}	3.2×10^{-8}	4.9×10^{-8}
^{239}Pu[c]	24,100 years	2.3×10^{-10}	3.8×10^{-8}	1.7×10^{-11}

[a]EPA classifies all radionuclides as Group A (known human) carcinogens. Radionuclide slope factors are calculated by EPA's Office of Radiation and Indoor Air. Ingestion and inhalation slope factors are best estimates (i.e., median or 50th percentage values) of the age-averaged, lifetime excess cancer incidence (fatal and nonfatal cancer) risk per unit of activity inhaled or ingested, expressed as risk/picocurie (risk/pCi). External slope factors are best estimates of the lifetime excess cancer incidence risk for each year of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil and are expressed as risk/year per pCi/gram of soil.

[b]A curie (Ci), the common unit of activity, is equal to 3.7×10^{10} nuclear transformations per second. (for example, 1 pCi = 10^{-12}Ci.)

[c]The toxicity constants for ^{239}Pu will be used for $^{239/240}$Pu. While ^{239}Pu and ^{240}Pu have different half-lives and external slope factors (but equal internal slope factors), only ^{239}Pu is listed here since 90 percent of the activity between the two isotopes is from ^{239}Pu.

Source: EPA, 1994.

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weight and time, and do not require corrections for gastrointestinal absorption or lung-transfer efficiencies (EPA, 1993).

External slope factors, which account for photon energy flux attenuation and buildup in soil, provide cancer risk estimates per unit exposure to a uniform concentration in soil. Because of the radiation risk models employed, both the internal and external slope factors are characterized as best estimates (i.e., median or 50th percentile values) of the age-averaged lifetime total excess cancer incidence risk per unit intake or exposure.

The dose conversion factors (DCFs) used for calculating dose are taken from Limiting Values of Radionuclide Intake and Air Concentration and Dose Conversion Factors for Inhalation, Submersion, and Ingestion (EPA, 1988) and are shown in Table 3-2. These DCFs are used to determine the committed effective dose equivalent (CEDE) resulting from intake of each radionuclide. The "committed dose" concept was introduced as a means of controlling occupational exposures to radionuclides that remain in the body for long periods of time.

DCFs are listed by solubility class and lung-clearance class for each radionuclide. Solubility classes are characterized by an "F1" value (Table 3-2). The F1 value represents the fraction of the radiological contaminant that is transferred from the gastrointestinal system to the blood. The F1 and lung-clearance class values for a particular radionuclide are dependent on the chemical form of that radionuclide.

The following subsections discuss important assumptions and procedures for internal and external exposure used to determine risks related to exposure to the COCs at OU 3. One factor that complicates evaluating radiation risk is that most of the data on health effects involve extrapolating from high-dose studies while accounting for the fact that most low-dose exposures are of the same order of magnitude as natural background.

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TABLE 3-2

DOSE CONVERSION FACTORS (DCFs) FOR INHALATION
AND INGESTION USED IN THE DOSE ASSESSMENT

	f_1^a	Inhalation Class[b]	DCF for Inhalation (Sv/Bq)[c]	DCF for Ingestion (Sv/Bq)[c]
^{241}Am	1×10^{-3}	W	1.2×10^{-4}	9.84×10^{-7}
^{239}Pu[d]	1×10^{-3}	W	1.16×10^{-4}	9.56×10^{-7}
	1×10^{-4}	--	--	9.96×10^{-8}
	1×10^{-5}	Y	8.33×10^{-5}	1.4×10^{-8}

[a]Fractional amounts of radionuclide absorbed across the gastrointestinal tract into the bloodstream.
[b]Lung clearance classification recommended by the International Commission on Radiological Protection (ICRP): Y = year, W = week.
[c]Sv/Bq = sieverts per becquerel.
[d]The toxicity constants for ^{239}Pu will be used for $^{239/240}$Pu.

-- = Not applicable.

Source: EPA, 1988.

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3.1 INTERNAL EXPOSURE

Internal exposure to radiation may occur through inhalation or ingestion of radioactive contaminants. Determination of risk due to internal exposure involves calculating the total amount of radioactive material taken into the body, and then applying an intake-to-risk conversion factor. The risk of cancer incidence from internal exposure to radiological contaminants was calculated using the intake to risk factors published in the annual 1994 HEAST (EPA, 1994).

The risk of cancer incidence from ingesting or inhaling radioactive contaminants is calculated by multiplying the total lifetime intake by the cancer-incidence risk factor for ingestion or inhalation. These slope factors relate risk of cancer incidence to intake of each radionuclide. The cancer-incidence risk factors are taken from HEAST (EPA, 1994).

3.2 EXTERNAL EXPOSURE

Radionuclides can have deleterious effects on humans without being taken into or brought in contact with the body. This is because high-energy beta particles and photons from radionuclides in contaminated air, water, or soil can travel long distances with only minimum attenuation in these media before depositing their energy in human tissues. External radiation exposures can result from either exposure to radionuclides at the site area or to radionuclides that have been transported from the site to other locations in the environment. Gamma and X-rays are the most penetrating of the emitted radiations, and comprise the primary contribution to the radiation dose from external exposures. Alpha particles are not sufficiently energetic to penetrate the outer layer of skin and do not contribute significantly to the external dose. External exposure to beta particles primarily imparts a dose to the outer layer skin cells, although high-energy beta radiation can penetrate into the human body.

Because measured radiation dose rates were not available, the HEAST risk factors (slope factors) for surface soil contamination will be used to calculate increased cancer incidence risks

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from external exposure. These factors assume uniform deposition of contaminants over a large area, which increases the uncertainty of such calculated risks.

3.3 EXPOSURE TO NATURAL BACKGROUND RADIATION

The health effects of radiation exposure are difficult to evaluate at low doses, partly because radiation is present naturally in the environment. The NCRP estimates that, on average, the background radiation dose to each individual is approximately 360 mrem/year (NCRP, 1987). Most of this dose is attributed to radon-222 (^{222}Rn) and its short-lived decay products.
Table 3-3 summarizes the average annual doses from predominant source contributing to background radiation exposure. Unlike many risks, the risks from exposure to naturally occurring background radiation are largely unavoidable. An evaluation of the risk from exposure to average levels of background radiation establishes a benchmark for judging the additional risk from anthropogenic releases of radionuclides to the environment. The distribution of the annual background dose to the U.S. population ranges from 75 mrem/year to 115 mrem/year for 80 percent of the population (EPA, 1990).

The risk of exposure to radon is calculated differently than the risk for other types of radiation exposure. The unit of concentration for radon is the working level (WL). The WL is defined as the concentration of radon daughter products in one liter of air that results in the emission of 1.3×10^5 million electron volts (MeV) of potential alpha energy. A working-level month (WLM) is defined as the exposure resulting from breathing air at one WL for 1 month (170 hours). The 200 mrem/year radon exposure discussed above equates to approximately 0.25 WLM/year.

The risk of radon exposure is calculated by multiplying the annual average exposure by the number of years of exposure (70.7 for a lifetime) and multiplying this result by EPA's radon risk factor of 360 fatal lung cancers per million WLM. This results in a lifetime risk of fatal cancer of 6.4×10^{-3}. In 1980, approximately 5 percent of all deaths were due to lung cancer. Thus,

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TABLE 3-3

AVERAGE ANNUAL EFFECTIVE DOSE EQUIVALENTS FROM IONIZING RADIATION FOR A MEMBER OF THE U.S. POPULATION

Source	Effective Dose Equivalent (mrem)[a]	(Percent)
Natural		
Radon	200	55
Cosmic	27	8
Terrestrial	28	8
Internal	39	11
Total Natural	294	82
Artificially Induced		
Medical		
X-ray diagnosis	39	11
Nuclear medicine	14	4
Consumer products	10	3
Other		
Occupational	< 1	< 0.3
Nuclear fuel cycle	< 1	< 0.03
Fallout	< 1	< 0.03
Miscellaneous[b]		
Total Artificial	63	18
Total Natural and Artificially Induced Sources of Ionizing Radiation =	357	100

[a]mrem = millirem or 1/1,00th of a rem.
[b]DOE facilities, smelters, transportation, and other sources.

Sources: NAS, 1990; NCRP, 1987.

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approximately 0.0064/0.05, or 13 percent, of lung cancer deaths could be attributed to background radon exposure (NCRP, 1987).

Two other categories of natural radiation exposure are exposure of lungs and of bone surfaces to naturally occurring alpha emitters other than radon. Values for fatal cancer risk for these categories are not shown because they are a factor of 100 to 1,000 less than the risks shown for low-LET and radon exposures.

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